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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

CONTENTS:

ALTERATION OF CEREBRAL FUNCTION IN MAN PRODUCED BY DIFFUSION RESPIRATION AND PROLONGED INHALATION OF CARBON DIOXIDE 35
Ewald W. Busse, Thomas M. Parry, Eli S. Goldensohn, Richard W. Whitehead, and William B. Draper

THERAPEUTIC DRAMA CLUB 41
Otto Schwarz

TREATMENT OF MENTAL PATIENTS WITH ANTI-DIURETIC HORMONE OF THE POSTERIOR PITUITARY 44
Lorant Forizs

SODIUM AMYTAL IN TREATMENT OF ANXIETY STATES 48
W. B. Cline, Jr.

NEUROLOGICAL CLINICAL PATHOLOGICAL CONFERENCE 53

PSYCHIATRY AND RELIGION 61
J. Dennis Freund

SANITARIA

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VOL. XIII FEBRUARY 1952 No. 2

Published monthly by
PHYSICIANS POSTGRADUATE PRESS
277 Broadway, New York 7, New York

EDITORIAL OFFICES
UNIVERSITY OF TEXAS SCHOOL OF MEDICINE
800 Avenue B., Galveston, Texas

Entered as second-class matter at the Post Office at New York, N. Y.

DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Alteration of Cerebral Function in Man Produced by Diffusion Respiration and Prolonged Inhalation of Carbon Dioxide

EWALD W. BUSSE, M.D., THOMAS M. PARRY, M.D., ELI S. GOLDENSOHN, M.D., RICHARD W. WHITEHEAD, M.D., and WILLIAM B. DRAPER, M.D.
Denver, Colorado

The effects of diffusion respiration upon the electrocorticogram of experimental animals have been reported in a previous paper.[1] The present report is primarily concerned with the electroencephalographic changes seen in human subjects during both diffusion respiration and prolonged inhalation of CO_2 under light pentothal sodium anesthesia. These two procedures are characterized by a marked and prolonged increase in the concentration of CO_2 in the alveolar air and blood. During diffusion respiration an excess of CO_2 accumulates endogenously as the result of absence of expiration, but during the inhalation of CO_2 the scource of the excess is obviously exogenous.

Diffusion respiration was originally described by Draper and Whitehead.[2] This phenomenon permits adequate oxygenation of the blood without respiratory movement if the animal has been denitrogenated and possesses an adequate circulation and a patent airway. When these conditions are satisfied and arrest of respiratory movement is induced by rapidly increasing the depth of pentothal sodium anesthesia, the animal continues to maintain adequate oxygenation of the blood and other tissues without artificial respiration of any kind. The physiological mechanism involved in the maintenance of oxygenation of the blood has been described in detail.[3] Due to the absence of expiration, the endogenously formed CO_2 accumulates to a high degree in the lung alveoli, and it was noted that the arterial concentration of CO_2 in dogs rose from an average of 36 Vol. % to 75 Vol. % during 45 min. of diffusion respiration and was accompanied by a considerable decline in the arterial and venous pH.[4]

The progressive changes seen in the electroencephalogram in 9 of the 10 dogs subjected to 45 mins. of diffusion respiration[1] were characterized by the development of periods of suppression interrupted by bursts of cortical activity similar to those reported by Swank and Foley who refer to the periods of suppression as "blackout." In our animals the bursts gradually decreased in both duration and amplitude until the suppression was apparently complete. This complete suppression continued for an average of 26 min. until the apnea was terminated by artificial respiration. In spite of the prolonged periods of cortical silence, the reappearance of continuous cortical activity quickly followed the return of respiratory movements and the consequent lowering of CO_2 tension.

From Division of Psychosomatic Medicine and the Department of Physiology and Pharmacology, University of Colorado Medical Center.

This investigation received financial support under a contract with the Office of Naval Research.

Pentothal Sodium used in these experiments was generously furnished by the Abbott Laboratories, Chicago.

Many investigators have been interested in the physiological effects of the inhalation of CO_2 and its possible therapeutic value in patients with mental illness. The use of CO_2 in the treatment of mental disorders was first decribed by Loevenhart, Lorenz, and Waters,[5] Leake, Guedel and Batsford[6] and later by Hinsie and co-workers.[7] Meduna's[8,9] recent studies on CO_2 therapy for psychoneurosis have renewed interest in the use of CO_2 in mental disorders. In some unpublished work using a technique similar to that of Meduna, we attempted to duplicate some of his encouraging reports of therapeutic success, but found that improvement which could be attributed to the inhalation of CO_2 was transient. However, as a result of this experience we then considered the possibility that prolonged inhalation of high concentrations of CO_2 or diffusion respiration, might be more effective in producing sustained improvement in personality functioning.

Method

Diffusion respiration was induced in three schizophrenic males who showed no abnormalities on physical examination, 31, 33 and 35 years of age. Each had been hospitalized for more than 3 years, and had received extensive treatment without improvement. The procedure was carried out three times with the 31 year old patient and once each with the others. EEG's were taken before, during and after the experiments. Two electroencephalographs were used, an eight channel and a four channel machine. Parallel studies included changes in venous pH, pulse rate, blood pressure, EKG, respiratory rate and plasma volume.

The patient was denied food for 15 hours, and fluid for 3 hours before the procedure. Morphine sulfate, grains 1/6 and atropine, grain 1/100, were given 45 min. prior to the induction of anesthesia. The technique of inducing diffusion respiration is as follows: Initially the patient is lightly anesthetized with pentothal sodium (thiopental sodium) I.V. and breathes 100% O_2 for 45 min. At the end of the denitrogenation period the anesthesia is abruptly deepened by the rapid injection of pentothal sodium until respiratory arrest ensues. The longest period of respiratory arrest maintained in man was 22 min., the shortest time was 11 min. 30 sec., and the average period, 16 min. 42 sec.

Dangers surrounding this procedure were anticipated and for this reason the responsible team included specialists trained in anesthesiology, internal medicine, neurophysiology, and neuropsychiatry. Special nursing care was provided throughout.

Forty-three periods of prolonged inhalation of CO_2 were carried out, usually at biweekly intervals, on 12 patients—7 males and 5 females. These patients ranged in age from 21 to 50. The clinical diagnosis of 10 of the patients was schizophrenia—7 paranoid and 3 catatonic. One patient was in a severe depression and the other was classified as a psychopathic personality with epilepsy.

Pre-inhalation precautions were similar to those outlined for diffusion respiration, except that morphine was omitted and one grain of sodium phenobarbital was given one hour before the procedure. Following the induction of light pentothal sodium anesthesia (first plane surgical) the patient breathed 100% oxygen for 15 mins. The shift to the desired concentration of CO_2 and O_2 was made slowly over a 5 min. period. After 15 min. at the maximum level of CO_2 another 5 min. interval followed during which the CO_2 content of the mixture was reduced until the subject was again breathing only oxygen. Six patients breathed the 20% CO_2 in O_2 mixture, 4 patients received 25% CO_2 in O_2 and two received 30% CO_2 in O_2.

Results

We have previously mentioned the progressive cortical electrical changes and apparent complete suppression of electrical brain activity in experimental animals during diffusion respiration. The phenomena also occur in human subjects. (Fig. 1.) During the period of denitrogenization typical continuous fast activity was seen in most instances but slower frequencies were present in a few records. In all of the patients when the anesthesia was deepened, a decrease in amplitude of the cortical rhythm as well as a slight slowing was observed. Respiratory arrest was followed by the development of alternating periods of suppres-

sion and burst of cortical activity. As diffusion respiration continued, the periods of suppression lengthened, the bursts of cortical activity became shorter and their amplitude decreased until no electrical activity remained even at maximum gain. Complete suppression occurred within 1 min. 30 sec., to 7 min. after respiratory arrest.

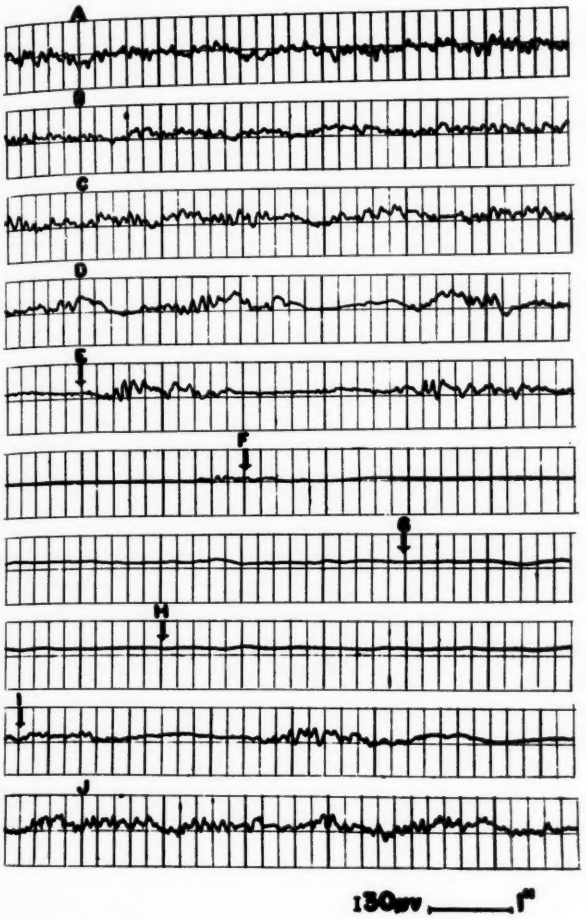

Fig. 1. EEG changes produced by different respiration.
A. detrogenation pH 7.46—light anesthesia
B. anesthesia deepened
C. minimal respiratory activity
D. first suppression ph. 728
E. respiratory arrest
F. 1'30" of diffusion respiration—last burt of cortical activity
G. 15' of diffusion pH. 7.19
H. 20' of diffusion pH 7.02
I. 7' post-diffusion ph. 7.18
J. Post-Diffusion Control

Shortly after the termination of diffusion respiration and the resumption of respiratory movements, bursts of cortical activity reappeared in a reverse pattern to that seen when disappearing and soon thereafter continuous activity returned. The time elapsed before the return to continuous activity was not proportional to the duration of respiratory arrest, and ranged from 9 to 31 min. This period of recovery was considerably longer than the relatively quick return of continuous brain activity which was seen in dogs following even longer periods of respiratory arrest. The EEG's of 2 patients taken from 1 to 3 days after diffusion respiration showed no evidence of change from the control. Three of the 5 periods of diffusion respiration were terminated by the spontaneous return of respiratory movement. In these 3 instances it was not possible to obtain venous pH at the precise moment of termination. However, the curves of venous acidity indicated that the pH was slightly above 7.0 at that time.

The post-diffusion course of all the patients was stormy and one patient expired 15 hours after the procedure. This patient had required a greater amount of sodium pentothal to induce respiratory arrest than needed by the others. His post-diffusion course appeared satisfactory until shortly prior to his death, when respiratory activity ceased and circulation failed. Autopsy findings did not contribute to the explanation for his death. In view of the obvious hazard, we felt we were not justified in continuing the work with diffusion respiration.

Prolonged inhalation of carbon dioxide and oxygen mixtures under light pentothal sodium anesthesia was a relatively safe procedure in our experience. In the group of 6 patients which received 20% CO_2 in O_2, 5 showed a gradual decline in amplitude of the cortical rhythm until at the conclusion of the 15 min. period of inhalation, the voltage usually had decreased to approximately 1/3 of the original amplitude. Apparent slowing of the rhythm which occurred seemed to be due to a loss of the faster frequency waves. Only one patient in the 20% CO_2 in O_2 group developed a typical suppression-burst pattern. In contrast to the 20% CO_2 in O_2 group all 6 patients to whom 25 or 30% CO_2 in O_2 was administered developed periodic suppression of the EEG. (Fig. 2.) However one patient in the 25% CO_2 in O_2 group failed to show periodic suppression in one of 4 trials, and in this one instance a gradual decline in amplitude was recorded. The amounts of pentothal sodium used could not account for

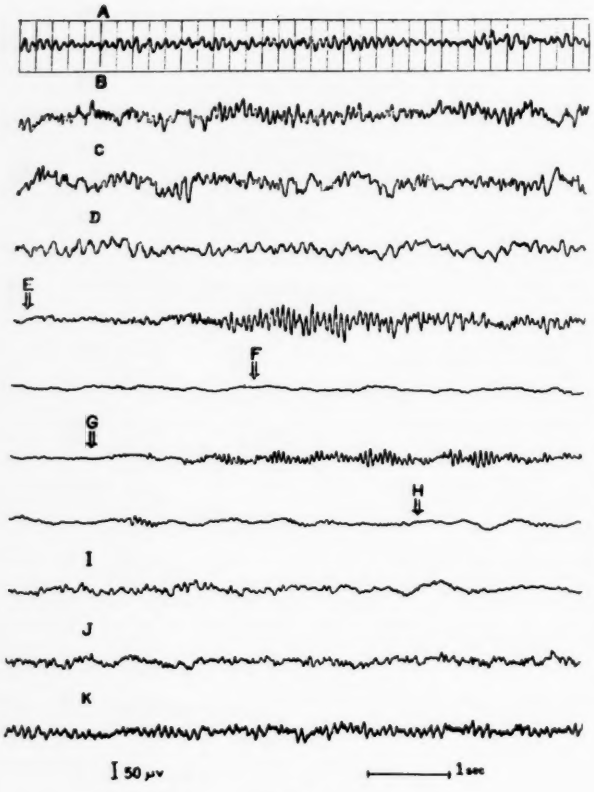

Fig. 2.

A. awake control
B. light anesthesia
C. CO_2 gradually introduced
D. 4' of increasing
E. 30% CO_2 + 70% O_2 inhalation mixture
F. 1' 30% CO_2
G. 2' 30% CO_2
H. 15' 30% CO_2
I. gradually decreasing CO_2 cone
J. breathing 100% O_2
K. 2' of 100% O_2
H. 15' 30% CO_2

the differences in the EEG change which occurred in the 20% CO_2 group on the one hand and the 25% and 30% CO_2 groups on the other. The venous pH changes offer a possible clue. The drop in pH was comparatively precipitous in the groups given 25 and 30% CO_2. At the end of 5 mins. of increasing the concentration of CO_2 to reach 25 and 30% the pH had fallen to an average of 7.00. Using 20% CO_2 under the same conditions the pH had fallen to only 7.33. After 5 min. on the full mixture the 20% CO_2 group had an average venous pH of 7.15 while the groups given the higher concentrations had an average pH of 6.98. No further significant decrease in pH (6.97 at 15 mins.) was found in the latter group, while the former continued to show a mild decline, reaching an average low of 7.08 at the end of 15 mins.

The periodic suppression which occurred in the EEG during diffusion respiration was similar to that seen during CO_2 inhalation. The duration of the periodic activity was variable, but in general, as complete suppression approached, the waves slowly decreased in amplitude and duration and the intervening periods of cortical quiescence lengthened. Initially a "burst" was usually composed of 12-18 per sec. waves, which quickly increased in amplitude. Later slower frequencies made their appearance, many of them in the range of 9-10 per sec., but some at 4-6 per sec. Small amounts of low voltage fast activity were occasionally seen in the second portion of the burst. After the slower waves became dominant, their amplitude gradually diminished and the record became flat. The bursts appeared simultaneously in all leads but were of highest voltage in the frontal leads, decreasing in the following sequence—frontal, motor, temporal and occipital.

The control EEG in three subjects was abnormal. One patient was an epileptic and her EEG was consistent with a convulsive disorder. The other abnormal EEG's probably resulted from courses of electro-convulsive therapy completed shortly before the patients were added to the CO_2 inhalation series. These two patients had normal EEG's before electric shock therapy. Although slow improvement in the post shock EEG's of both these patients would be expected, rapid striking and improvement in the brain waves occurred immediately after the first CO_2 inhalations.

During 30% CO_2 in O_2 administration to one schizophrenia patient after an almost complete disappearance of burst activity, a fast frequency appeared. (Fig. 2.) This activity was characterized at the onset by frequency of 14-20 per sec. at very low voltage. The amplitude was reached at 15 mins.

In all instances the inhalation of the CO_2 mixtures produced a marked increase in the pulmonary minute volume without significantly changing the respiratory rate. Occasionally, the EKG revealed transient extrasystoles. There was a moderate rise in heart rate and blood pressure during the inhalation of CO_2. Blood pressure fell below control levels shortly after breathing of CO_2 was

resumed. The fall in blood pressure was readily checked by small amounts of neosynephrine intravenously. No convulsion occurred during the procedures. During CO_2 inhalation plasma volume showed a decrease of 10.2% by the dye (T1824) method and 10.5% by changes in protein concentrations.

Clinical Results

Of the three patients who had been placed in diffusion respiration one improved, one showed no improvement and one died. The patient who had been subjected to diffusion respiration three times showed alleviation of his catatonia and was moved to an open ward where his adjustment was satisfactory. Of the twelve patients given prolonged CO_2 inhalation, four improved sufficiently to be permitted to return to their homes. The remainder required continued hospitalization. Immediately after treatment transient increase in communicability, which was relatively free of psychotic manifestations, was frequently observed.

Discussion:

This work has shown that diffusion respiration occurs in human beings deprived of respiratory movements. Draper and Whitehead first showed the existence of this phenomenon in dogs in 1944. It is probable that diffusion respiration functions as an ancillary mechanism during normal breathing.

It has now been shown that the prolonged electrical silence of the brain, which was first seen in animals during diffusion respiration is also encountered in man, and that this prolonged absence of electrical activity is apparently a reversible process. Other investigators, particularly Swank and his co-workers,[10] have seen the complete disappearance of brain waves in experimental animals under very deep barbiturate anesthesia and infer that such a development heralds approaching death. Van der Horse[11] reported that rabbits in profound ether anesthesia show progressive changes in the EEG until "all oscillations disappear and the EEG appears as a straight line," and that upon the lightening of the anesthesia the activity returned. From these reports it is apparent that the deep coma due to barbiturates or ether is associated with electrical silence in the cortex. However, we believe that the changes in the EEG we observed were the result of the combined action of the sodium pentothal and CO_2. The prompt return of periodic cortical activity during the time CO_2 was being blown off at the termination of diffusion respiration is strong evidence that CO_2 played a considerable role in maintaining electrical silence.

The fact that suppression burst activity occurs during prolonged CO_2 inhalation and does not occur during light surgical pentothal sodium anesthesia without CO_2, is evidence that CO_2 plays a role in suppression-bursts. In this connection it is interesting to note that although Swank and Foley ascribed no particular significance to this fact, the blackouts described by them did not occur until the arterial blood CO_2 had been raised by an average of 10 vol. %. Swank and his co-workers[10] have also shown that blackouts appear in deep barbiturate narcosis but do not occur in ether anesthesia. Our work reveals that individuals inhaling a mixture of 20% CO_2 have a gradual decline in wave amplitude which is supposedly characteristic of ether anesthesia, and that suppression-bursts readily appear when the concentration of inhaled CO_2 is increased to 25%. 30%.

The manner in which the marked increase in the quantity of CO_2 in the blood and tissues and the corresponding decreased pH influences the functioning of the brain is not known. Dusser de Barenne, McCulloch and Nims[12] have shown that a fall in pH of the brain cortex produced by the intravenous injection of hydrochloric acid is accompanied by a reduction of spontaneous cortical electrical activity. Lubin and Price[13] found that nearly a fatal amount of acid was required to produce an effect comparable to that of small rises in CO_2 tension and for this reason contended that the reduction of pH is not the only mechanism through which CO_2 alters the EEG. Lorente deNo[14] reported that CO_2 increased threshold of peripheral nerve by increasing its membrane potential and that this effect is not due to pH change. Brooks and Eccles[15] observed that the inhalation of CO_2 causes a depression of spinal neurons and increases synaptic delay. Burge et al[16] showed that 100% CO_2 causes the

brain to become electro-positive in reference to the sciatic nerve and other elements of the neural axis. Goldenson, Schoenfeld and Hoafer[17] found that 20 and 30% CO_2 in O_2 increase the positivity of the brain markedly and that these in voltage are of considerable greater magnitude than those found with light or moderate pentothal anesthesia. These isolated findings only indicate the complexity of the mechanisms involved in the functional changes seen with high concentrations of CO_2.

In view of Meduna's theory of the action of CO_2 in psychic disturbances and the conception on our part that the electrical silence may in some way break up long term memories which contribute to the psychosis we felt that the diffusion respiration procedure with its apparent complete suppression of cortical activity and the safety of its use in animals warranted clinical trial. The fatal outcome in one case, however, halted our efforts in that direction.

The prolonged inhalation of CO_2 in concentrations up to 30% under light pentothal anesthesia appeared to be a relatively safe procedure. The beneficial effect of prolonged inhalation of high concentration of CO_2 in patients with schizophrenia is not sufficiently sustained to appear to be of appreciable therapeutic value. We did however again observe, as have others in the past, the transient therapeutic effect of the inhalation of CO_2.

Summary and Conclusions

(1) The phenomenon of diffusion respiration enables human subjects to oxygenate their tissue and continue to live during prolonged absence of respiratory movements. This procedure is associated with considerable danger to the patient.

(2) Prolonged administration of a combination of pentothal sodium anesthesia with inhaled mixtures of 20%, 25% and 30% carbon dioxide can be used in man to lower the pH of the venous blood to slightly below 7.00 and is apparently a relatively safe procedure.

(3) The development of periodic suppression or "blackouts" is a feature of the EEG changes seen in both diffusion respiration and during inhalation of 25 and 30% CO_2. The periodic suppression is probably due to the combined effect of the pentothal sodium and high concentrations of the CO_2.

(4) Complete suppression of the human EEG occurs for prolonged intervals during diffusion respiration. This suppression is apparently reversible.

(5) Conclusions are not justified as to clinical value. It can only be stated that 4 of 12 patients improved following CO_2 inhalation, and that one of 3 patients improved following diffusion respiration.

Authors' Note: The authors gratefully acknowledge the assistance of Miss Julia Kassanchuk, R.N., of the Anesthesia Department of the University of Colorado Medical Center who contributed her services to the research project.

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Therapeutic Drama Club

OTTO SCHWARZ
Palo Alto, California

From September 1950 to June 1951 a special form of dramapsychotherapy was attempted with a group of forty epileptic patients at the Cambridge State School and Hospital, Cambridge, Minnesota.

Epileptics with grand mal and psychomotor seizures and a few non-epileptic higher grade feebleminded patients were selected on the basis of their professed interest in dramatic activities. Both sexes were represented in almost equal numbers and the age distribution ranged from fourteen to about thirty-five, with one patient having passed her fiftieth birthday.

Under the direction of the writer, a group of students from the University of Minnesota explained to these patients the need for and the advantage of an active drama group within the Cambridge community. The patients were invited to join with the student volunteers in the formation of such a group. In October 1950, with patients and volunteers enjoying equal membership status, the "Cambridge Drama Club" was founded. From the very beginning it was stressed that the proposed activities would satisfy not only a need of the patients, but of the volunteers as well. Words such as psychotherapy or "treatment" were not to be employed under any circumstances. Thus, patients began to identify with the group, and to consider themselves integral parts of a functioning social organization. "Belonging" to such an organization and being a vital part of it, was a new experience for many patients.

It was suggested to the group that they write their own skits and plays rather than selecting material from published works. The majority of patients manifested apprehension at the thought of writing plays; they lacked the necessary self-confidence to put their imagination or experience into writing. This apprehension did not diminish even after they were advised perfection was not expected, but merely simple attempts to put on paper their past experiences. To alleviate their anxiety, it was decided that the actual writing would be done by the volunteers, while the acting would be left to the patients. However, to assist the volunteers in writing these plays and skits, each patient was requested to address a letter to the writer of this paper, outlining the type of role he would like to enact on stage. It was pointed out to the group that this procedure would assure each club member of being assigned interesting and desirable parts.

The patients responded very favorably and a letter in one form or another was written by each club member. A great many of these letters were indicative of emotional difficulties. One girl desired to play the part of a maid "who is always being scolded by nasty, rich society people." Another girl wanted to play the part of a naughty youngster "who drives everybody crazy by mixing up gift cards attached to Xmas packages." A male patient desired the part of an aggressive, successful businessman "who treats

From Dept. of Psychology, Stanford University.

his employees in a nasty way." These are only a few examples, but aggression, whether directed outward or turned upon the self, constituted a frequently encountered phenomenon. Equally pronounced and commonly recurring were anxiety states and manifestations of intense guilt feelings—frequently a result of religious conflicts.

It became the task of the volunteer group to coordinate the various, and often unique, parts requested by the patients and to fit them into coherent plays. Instead of finding actors to fit the characters, here was a situation where the play was being written to fit a number of principals. Since emphasis was placed upon utilizing the talents of everyone of the forty club members at all times, it became necessary to write as many as six or seven short plays for a single evening's performance.

To prepare for this performance, the club members were divided into sub-groups of five or six each, depending upon the number of parts in each play, and placed under the 'technical' direction of one of the volunteers. The volunteers directed the production of the plays, and, briefly, treated the patients much as any director would treat his actors. The patients had requested at the outset that mistakes or errors in interpretation be pointed out and the volunteers readily complied with this request. It was observed that indiscriminate approval of even inferior performances tended to decrease the value of this approval and an effort was made at all times to maintain an attitude of honesty. If a patient's efforts warranted approval, he would be complimented. If, on the other hand, it became obvious that certain patients were not cooperating with other members of their group and displayed no effort to raise the quality of the production, they were called aside and efforts made to learn the reason for their uncooperative behavior. Often the cause for such uncooperative behavior centered in a private feud between two patients within the same sub-group, and in such cases the problem was solved by assigning one of them to another sub-group. Incidences of uncooperative behavior, however, were rare, and the majority of patients manifested a fine spirit of cooperation.

Ordinarily, three or four rehearsals were required to insure an adequate stage performance. At the final peformance, the audience consisted of fellow patients and staff members, and every attempt was made to fill the auditorium. There were times when the 'Drama Club' performed before audiences of more than five hundred fellow patients, staff members and interested persons from St. Paul and Minneapolis.

The first full dress performance, taking place approximately one month after the organization of the drama club, was preceded by a great deal of stage fright. Minutes before the curtain was about to rise, those patients exhibiting behavior indicative of intense anxiety feelings were informed that they would not be obliged to perform unless they really wanted to. If they so preferred, volunteers would assume their parts. This action was prompted by an unwarranted fear that the prevailing anxiety among the patients might lead to numerous seizures. None of the patients availed themselves of this opportunity to withdraw, and not one single seizure occurred throughout the entire evening.

In order to increase the patients' security and self-assurance, and to prevent the possibility of traumatic experiences resulting from suddenly forgetting lines, two volunteer workers acted in the capacity of prompter. Upon a pre-arranged signal on the part of the patient, the volunteer nearest to that patient would read o him his next line.

Immediately upon completion of the last play, and while the applause, literally speaking, was still ringing in the patients' ears, they were assembled and congratulated upon their excellent performances. The existing conditions of pleasurable excitement were utilized to provide the transition for the next step in the program. The writer of this paper addressed the patients and stated that while the acting had been accorded the audience's unanimous approval, the construction and content of the plays had left much to be desired. The patients were requested to give this matter consideration and to offer concrete suggestions on how to improve the quality of the plays both as to content and performances. Since the patients were, of course, aware that the vol-

unteers had written these 'poor' plays, an awkward and somewhat embarrassing silence ensued, and the matter was allowed to rest.

Our next weekly visit to the hospital produced interesting results. Three patients, somewhat apologetically, reported that they had had some ideas about plays, and rather than simply tell us about them and leave the actual writing to us, as had been done in the past, they had themselves written the plays. As already mentioned, their manner was quite apologetic and they were obviously afraid of offending.

The plays were read and the patients informed that their work would be included in the next production. During our subsequent meeting six more plays were turned in, and within a few weeks, twenty-two of our forty patients were writing plays about their own experiences, hopes and emotions. To further their interest in this work and to provide them with an added means of self-expression, the authors were assigned the responsibility of directing their plays. They were further encouraged to choose the cast for their plays. In cases of conflict, two authors wanting the same individual for their plays, the problem was solved by group discussion. Each rehearsing group had assigned to it a volunteer worker who was instructed to in no way interfere with the director in the production of the play. The volunteer's duty was strictly advisory. He would allow the director to proceed on a trial and error basis and would offer advice only when specifically requested. Thus with the advent of patients writing their own plays, a form of almost completely non-directive therapy was adopted. The 'play,' from inception in the form of an idea to completion in the form of the actual performance before an audience, became entirely the patient's responsibility.

While no recognized devices of personality measurement were employed, a majority of the patients exhibited remarkable behavior changes in the course of this program and certain trends could be definitely noted. Firstly, an appreciable increase in self-confidence could be observed. Patients who had been terrified at the idea of performing before an audience, and had requested non-speaking parts only, commenced to demand larger and more important roles requiring their presence on stage for longer and longer periods of time: such patients actually felt slighted when assigned a supporting part.

Release from emotional tension as a result of the drama therapy became evident in a number of cases. The patient who had requested the part of a maid scolded by her employer, requested to be assigned the role of a kind and permissive lady. When given such a part, however, she would speak permissive lines in an obviously aggressive manner. Here probably was a case of inwardly turned aggression directed into external channels, brought about partly by the lack of reward accorded her impersonations. The audience would enjoy and applaud aggressive impersonations and accord little, if any, sympathy to parts such as had previously been requested by this patient.

Lastly, the program provided the patients with a much desired constructive activity. It took many hours of pleasant effort to plan, write and enact these plays. Hours that might have been spent brooding about other things were occupied with constructive thought processes. The various opportunities for self-expression provided a strong motivational force; a force sufficiently powerful to induce a female patient, five minutes after undergoing a moderately severe grand mal seizure, to go on stage and perform.

As regards the permanent effect of a "Therapeutic Drama Club" on epileptic seizures, it is only possible to speculate at this time. Since it was impossible to keep constant factors such as anti-convulsive drug therapy or dietary restrictions, the program cannot statistically be credited with any actual reduction in seizures. It would only be fair, however, to assume that the stimulated brain activity coupled with the pleasurable excitement produced by performing for an audience may well tend to reduce seizures in many individual cases. An equally important consideration is the educational value of such a program to the hospital personnel, the friends and relatives of the patients, and the public at large.

Treatment of Mental Patients With Antidiuretic Hormone of the Posterior Pituitary

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After the almost complete nihilism that prevailed in the fields of therapy for schizophrenics, a new era started with the introduction of the different shock therapies. Various types of shocks seemed to produce clinical improvement without our knowing even in approximately satisfactory terms what the schizophrenic process is or what the mechanisms of these shocks were. For a considerable time it was believed that some biological antagonism between schizophrenia and epilepsy might be responsible for the apparent clinical improvement. The significance in the changes of the water metabolism in epilepsy was already known. In 1936 I started experiments believing that overhydration with great doses of water and Pitressin might produce epileptic convulsions in a schizophrenic, and thereby a different way of treatment could be established for schizophrenias.

Data in world literature about the physiology of schizophrenia appeared to be encouraging; the findings of Hoskins and Jellinek in 1935 showing an increased turnover in the body water of schizophrenics indicated that this way might even lead to a point closer to the etiological line. A preliminary report was made in 1937 on the theoretical possibilities of these investigations. Adverse circumstances in Europe prevented any extensive research and only sporadic observations were made which showed that no epileptic convulsions occur under this regime in a schizophrenic; however, clinical improvement followed in some cases.

We are all aware of the traps in the research field in the treatment of schizophrenia and of the many disappointments that followed the mushrooming treatment methods. Therefore, it was my aim to find proofs in a long-range project and in a number of cases which might have statistical significance indicating that something happens in a schizophrenic mind while the patient is under this treatment. Our observations in this group of patients treated with the hormone of the posterior lobe are aimed to register those facts that might be interpreted as non-coincidental change in the course of a mental disease, trying to exclude mistakes in the sense of "post hoc, ergo propter hoc." We also tried to exclude the possibility of changes on account of altered environment (transfer to treatment ward), or perhaps to take into consideration separate evaluation of these factors.

From North Carolina State Hospital, Butner, N.C.
Read at the Annual Meeting of the American Psychiatric Association in Cincinnati, Ohio, May 9, 1951.

Material and Methods

Our cases were selected from a population of approximately 1500 in a hospital that had been in operation for only three years and which received overflow patients by transfer from other state hospitals, particularly patients who had not been able to improve over a period of five to ten years' hospitalization—cases that had been abandoned by their families and showed a more or less constant downhill course and seemed doomed to a permanent custodial status. The experimental cases were selected from the numerous so-called "ward problems"—fighters, soilers, and the destructive ones.

Out of a number slightly over one hundred, 80 cases can be more or less fully evaluated by this time. Out of the 80 we had only three acute cases from our new admission ward---all of them schizophrenias—and the rest consisted of 62 schizophrenia, nine mentally defective with a superimposed schizo-form psychosis, two chronic manias, two seniles, two involutionals, and three post-encephalitic conditions with psychotic signs but without Parkinsonian syndrome. Our evaluation was focused on the group of schizophrenias and only approximate opinions could be formed about the minor groups.

The method of treatment consists of daily injections of ten units of Pitressin (Parke-Davis) for the first ten days. After this, ten

units are given every second day. We make very few deviations from this general pattern; however, in one or two cases we have tried to speed up the relapse period by resuming the daily schedule, and in numerous cases we tried to go over a third or fourth day schedule, but it did not seem to be sufficient, and we saw not only a slowing-up but even relapses which subsided again after resuming the two-day or daily schedules. The patients were not given any special diet; their fluid intake was slightly encouraged.

About ten or 15 minutes after receiving the injection, the patient becomes pale. As a rule there is no subjective discomfort associated with this; no fainting nor dizziness occurred in any of our cases, but within 20 minutes after receiving the injection most of the patients had an imperative urge to urinate, and in many instances, one or two stools occurred, but only rarely was this associated with any abdominal discomfort. Women continued injections during menstrual periods; apparently they had little additional discomfort.

Blood pressure readings were made before and about 25 minutes after Pitressin was given and no substantial changes were seen. One would have expected a rise of blood pressure readings, but in many cases, paradoxically enough, there was a drop of ten or 15 millimeters.

During the whole year of experiments including more than 100 patients we had one activation of a tubercular process and we do not know whether the Pitressin treatment had any responsibility for the activation. Interestingly, after a few days of elevated temperature, which actually gave rise to the further tubercular examination, the Pitressin was stopped, and the next day the reaction immediately disappeared without any improvement, of course, in the general condition. No other ill effects whatsoever were observed.

Results

Trying to describe the course of events during this treatment, I would mention as first the increase of sleeping time. Almost without exception patients appeared to sleep better at night, and most of them would also doze in the daytime. Those who were in contact sufficiently to tell us about their subjective experiences made observations that they were more relaxed, sometimes tired and wanting to sleep. According to them their sleep was deeper, more refreshing and more satisfying. Some patients appeared drowsy, spending most of the day and the night in bed as though they were under sedatives, although with the exception of a very few emergency occasions, the use of sedatives was completely excluded during the experiments. This effect of the Pitressin could usually be noted with the first day injection.

As a continuation of this relaxing and sedative effect, there was a general reduction in psycho-motor activity. Highly combative and destructive patients showed a marked change at the end of the first week of treatment. In exceptional cases this initial improvement was delayed until the end of the second or third week. After a few days of improvement, a relapse followed. During this relapse period most of the original symptoms were reactivated and in chronic patients the level of relapse reached at least that of the starting period of the treatment, and very often it went even further so that early symptoms of their psychosis which had not been observed for many years during their stay in the hospital and that were known to us only through clinical notes came to the surface again. We might say that a reactivation of the entire psychosis took place, and long-forgotten behavior patterns were shown; old hallucinatins reappeared; former combativeness returned; regression to oral and anal levels developed. Very frequently elderly female patients started to masturbate, a symptom which had not been observed in them for several years. Towards the end of this phase, perhaps as a first sign of the more permanent improvement, there was a growing adequacy of the emotions.

The general emotional flatness in simple schizophrenia cases seemed to alter. Patients who had not been seen crying for several years became overly emotional. Autism slowly subsided; awareness of the environment increased. Excited patients were able to spend more and more time in the day rooms or porches, the hours of seclusion becoming rarer. Toilet habits became tidier. Spoon feeding became unnecessary and there

was a general improvement in eating habits.

Improvement was evidenced in the reduced number of injury sheets and escape reports. So-called "ward problems" disappeared. There was no improvement in destructiveness when it was confined to clothing and bed linen as an isolated symptom. Patients were kept in the same ward, and the ones who did not go to occupational therapy before seemed to develop an interest in this from the other patients and asked if they could be permitted to join the group. Interest in hospital recreational facilities grew and in some cases was also promoted by coaxing. It is interesting to note that frequently visiting relatives discovered an improvement in the patient two or three weeks earlier than we did—the patients showing definitely more interest in their families, asking questions which had not been asked for years, requesting clothing and in some cases patients spontaneously started to write home. We might quote one of our cases, a middle-aged man who had not written home for sixteen years, who started correspondence with his family without their asking him to do this. During this phase the hallucinations slowly faded away and the patient's insight increased.

A constant finding was a substantial increase in body weight, sometimes as much as forty pounds within four to eight months, usually running a parallel course with mental improvement, following its ups and downs, fairly closely. Another collateral observation was that our patients showed a more violent but definitely shorter course during an upper respiratory tract infection. The significance of both of these findings might have some role within the framework of the general adaptation syndrome.

Slightly less than one-third of these patients did not show any substantial change during the course of the treatment. One out of the total of 80 seemed to have undergone more deterioration. Six of them showed temporary improvement, maintained only for a few months then relapsing to their original level. Twenty showed slight but noticeable improvement which was maintained; in nine other cases the improvement was significant and unmistakable. In three cases the improvement reached the point where the patients could be sent home and out-patient treatment continued, but for technical reason this cannot be carried out up to the present time. Another four patients became well enough to go home, continuing their treatments at home, receiving ten units of Pitressin three times a week. The home stay of these patients has extended over many months.

I can mention one significant illustration of the value of this treatment. A female schizophrenic patient had been in hospitals four years but had a history going further back. Under treatment she recovered and returned home, but after three weeks refused to go regularly to her family physician for the Pitressin injection. She relapsed immediately and her husband wanted to return her to the hospital. I persuaded him to have her physician resume the injections. She improved immediately and is still at home.

The speed of improvement, the timing of the initial improvement, the relapse period, and the final improvement seem to depend on the previous duration of the psychosis. Acute cases show their initial improvement four or five days after the start of the treatment, and their relapse period lasts for only three or four weeks; whereas long-standing cases show the initial improvement phase after only ten or fifteen days, and the relapse phase is drawn out over a period of three or four months. It is almost a rule in prognostication of a case that if there was the slightest initial improvement during the first two or three weeks, we could always expect improvement after the expected relapse period. Cases that do not show this initial improvement very seldom show any substantial remission.

The second minor group of the so-called mentally defective included our only case that seemed to get worse, and only temporary significant changes in seven. Response was poor in four other cases—two pairs of siblings where apparently a serious hereditary factor is present. The post-encephalitic group had not changed any; neither did the manics; whereas the involutionals showed at least temporary or slight changes, and two senile patients improved to a significant degree. Perhaps the most significant improvements occurred in the simple

form of schizophrenias. Significantly enough, catatonias did not take first place in this series.

Discussion

In the light of the general adaptation syndrome described by Selye these experiments may have a different significance from my first hypothesis. Quoting Selye:

"It becomes increasingly more evident that many of the time-honored though not spectacularly effective non-specific therapeutic measures such as fever therapy, shock therapy, parental administration of foreign proteins, blood-letting or near starvation are beneficial largely through the general adaptation syndrome. Often their principal value appears to be that they stimulate ACTH and glucocorticoid production. Whenever this is so, it may be preferable in the future to inject ACTH or cortisone. In certain instances, however, non-specific therapy proved efficacious where ACTH failed. For instance, with the mental patients who did not respond to ACTH it could be shown by subsequent shock therapy that they had been benefited by non-specific stress. Here, presumably, certain specific changes incident to the shock, act as conditioning factors of the hypophysis adrenal discharge, or separately through other channels of the general adaptation syndrome for instance, the nervous system or catabolic response of the alarm reaction."

"There is much to suggest that at least certain types of stress for instance, emotional stimuli, can increase the production of antidiuretic hormone which is probably identical with vasopressin. This effect is undoubtedly mediated by nerve tracts descending from the hypothalamus to the posterior lobe since, unlike the discharge of the ACTH, it is abolished by trans-section of the hypophyseal stalk."

The work of Altschule and his co-workers has shown that in certain cases, at least, antidiuretic hormone production and consequent changes in the water metabolism occur during or after electric shock.

Our experiments together with the above quoted references indicate that emotional stress has a more or less specific place within the general adaptation syndrome and that its channels are somewhat different from those of other types of stress. Research work on emotional stress related to the general adaptation syndrome has shown that mental patients, mainly the schizophrenic group, respond differently from the response of the normal group. The spectrum of this response, or at least a certain sector of it, has been analyzed extensively. Significant changes have also been registered in the water metabolism, but no comments were made as to the mechanism of these changes of the water economy. The anterior pituitary and the adrenal cortex are considered in publications. There was scarcely a reference made to the posterior pituitary which in this specific case of emotional stress might have a more pronounced significance than the other two groups of hormones.

Unfortunately, our hospital is not equipped for any of those complicated laboratory investigations, but it would be highly desirable for research centers adequately equipped to turn their attention in this direction. Besides the importance of this question in cases of mental disease, we may perhaps mention another field where its significance might be great and this would be the mental stress aspect in military personnel as well as civilian; possibly flying and submarine crews might be benefited more than the ordinary soldier on the battlefield.

On purely psychiatric terrain we are setting up new groups of patients in whom combinations of Pitressin with electric shock and insulin shock will be investigated. From the very few experiences that we have along this line it seems to us that Pitressin cases, when combined with electric shock, respond with a more violent convulsion under a lower voltage and time factor. Many cases have shown improvement at an earlier date than with electric shock alone. In some cases two or three shocks were sufficient to bring the patient to a level of substantial improvement; in other cases where intensive shock therapy did not seem to help the patient, after a few days of mixed regime, a substantial improvement could be seen. Other cases which did not respond in any significant degree to Pitressin treatment alone showed rapid improvement after two or three electric shocks.

The time has been too short, as yet, to make any statement about how the continued Pitressin treatment, even after the remission, might prevent relapses or prolong the improved functioning level. We might still mention that this treatment alone prevents the complications of accidents to bones and joints that may occur in shock treatment.

Sodium Amytal in Treatment of Anxiety States

(A Preliminary Report)

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The problem of therapy in anxiety states has become more and more important in the everyday practice of medicine whether it be seen from the viewpoint of the general practitioner, the psychiatrist or of other specialists. It is our purpose to describe a new method of administration of a well known drug which we have found to be effective, in conjunction with psychotherapy, in the treatment of milder anxiety states. We can report excellent results in intravenous administration of Sodium Amytal, not, however, in the conventional manner in which it is employed in narcosynthesis.

This therapeutic method was developed independently but was originally suggested in an excellent monograph by Susselman, Feldman and Barrera.* These writers used intravenous Sodium Amytal in average doses of 1½ grains injected over a period of about one minute, primarily for the purpose of differential diagnosis between organic disorders and somaticized anxiety symptoms. They conclusively demonstrated the value of Sodium Amytal used in this way. They classified tension symptoms in four groupings: musculo-skeletal (by far the largest group), gastro-intestinal, cardiovascular and genito urinary, and showed that the relief of discomfort in anxiety states far exceeded and outlasted the expected analgesic properties of the drug.

Our experience fully confirmed these findings. We felt, however, that such a valuable diagnostic test could surely be utilized to support conventional psychotherapy. We found, by cautious trial over a long period, that Sodium Amytal given intravenously after the therapeutic interview in relatively large doses injected very rapidly served to relieve the symptoms of anxiety for significant and increasing periods of time and has proved to be a very effective addition to the psychotherapeutic armamentarium.

*Intravenous Injection of Sodium Amytal as a Test for Latent Anxiety. *Arch. Neurol.-Psychiat.* 56:567 (Nov.) 1946.

To describe the method employed, we shall report here the instructions given to physicians in out-lying communities for administration of Sodium Amytal according to our therapeutic plan where the patient was unable to come to Corpus Christi for treatments.

Regime of Therapy

The patient is given the intravenous injection while reclining, having been informed that he will experience a dizzy or drunk-like sensation and usually a bitter metallic taste a few seconds after the injection is completed. Both of these sensations last only a few (3 to 5) minutes and the patient will be able to go home in ten minutes if he wishes. If convenient, he may be allowed to rest for a longer period and may fall asleep after injection, but can easily be awakened.

The injection is given in dilution of 1 cc of sterile distilled water to 1 grain of Sodium Amytal, the solution having been mixed in the Sodium Amytal ampoule. (This dilution is chosen for convenience in measuring dosage.) The injection is made rapidly; that is in about 10 seconds. The average initial dose is 2.6 gr. (2.6 cc) though this may be reduced for smaller persons and increased for those habituated to barbiturates. A convenient means of measuring the speed of injection and of attaining a standard speed of injection is to inject the solution just about as rapidly as possible with a 25 gauge needle. The second and subsequent doses may be increased to 2.8, 3.0 and 3.2 or more grains if necessary to produce the desired relaxation.

The speed of injection may seem excessive but is essential to the success of the treatment. No instance of respiratory depression has been noted in thousands of such injections in doses less than 3.75 gr. (see literature with package ignoring the stated precaution concerning speed of injection).

The interval between injections is increased each time if possible. The usual intervals in the mild to moderately severe

anxiety state is 5, 7, 10, 12, 14, 21 and 30 days. These may have to be shortened in more severe cases and may be lengthened in cases where the response is better.

In so providing general practictioners with instruction in this therapy, we deliberately intended to test the efficacy of the drug alone, without conventional psychotherapy. Suffice it to say, it did not work. Patients reported that treatment so given helped little if at all.

Many of our colleagues may be exceedingly skeptical concerning the value of this method, and indeed we have delayed submitting this article for publication because we ourselves wanted more corroboration. However, five years of experience with this treatment in thousands of cases has convinced us that we should present our findings for critical evaluation by others. We recommend this method only for competent psycho-therapists. Unfortunately our experience has shown that others will attempt, without success, to employ the method, depending solely on Sodium Amytal for beneficial results.

It is to be noted that the injection of Sodium Amytal is given after the therapeutic interview. This plan was adopted in order to avoid the possibility of confusing the drug effect during the interview with possible narcosynthesis-like effect upon the interview. Furthermore, we found that the patient was not much interested in discussing his problem after he had been relieved of his symptoms.

Upon completion of physical and neurological examinations and study of all reports submitted by the referring physician, the patient is advised of the probable relationship of organic and functional factors in his illness. He is assured that the use of the term "functional" does not mean that he only imagines that he has pain, but that it means that his symptoms can be equally disabling without organic or structural change in the body; that excess of nervous tension can and does produce an infinitely wide variety of symptoms, many of which seem to the patient to be organic in nature. It is important that this point be made clear early in treatment since the patient has usually been told that there is nothing wrong with him, that all he has to do is "pull himself together" or that he is just nervous and nothing can be done about it. When it is explained that his illness is none the less real for all the fact that physical and laboratory findings are negative, and that something can be done about it, then the patient begins to feel that possibly, at long last, he may receive some help.

Another point which we have found important to explain to the patient early in the treatment is the fact that the anxiety states have nothing in common with mental illness and that, even if he has felt that he might lose control or "blow his top," he is in no danger whatever of "losing his mind." Usually the patient does not inquire about this at first because he is afraid of the answer much as persons who suspect cancer refuse to be examined. Even though he has not inquired, he is usually visibly relieved to be told that there is no danger of insanity. With these two essential points covered the patient is usually ready to listen to almost anything the doctor has to say and is distinctly relieved that he is not getting the usual brush-off or "pink-pills-for-pale-patients" routine.

We tell him that we believe many or perhaps all of his symptoms are caused by excess or nervous tension and we should like to perform a simple test which will indicate whether or not our opinion is correct. It is explained that a small intravenous injection of Sodium Amytal will relieve his tension promptly and that all symptoms caused by excess of tension will be relieved or greatly ameliorated. Many patients ask if we are giving "truth serum" to make them talk while unconscious. Whether or not they ask, it is explained to all patients that they will be given a small dose which will cause no loss of awareness but which will make them dizzy for two or three minutes and cause a bad taste; that after ten minutes they will be able to leave the office and drive a car in perfect safety or that they may remain for a longer time if desired and may even sleep for a short while if they wish to do so. Another point that is mentioned, whether inquiry is made or not, is that this procedure is not habit forming. With this preparation the patient is usually willing and anxious to

proceed with the test. He is told that should the test be positive (i.e. relief of symptoms for a period of two to four days following the injection) then the same injection can be used in the treatment of his illness, of course in combination with the usual psychotherapeutic procedure.

It has been found helpful in explaining the nature of the illness, which is always difficult, to use a simple analogy which seems to clarify the problem. We refer to the similarities between nervous tension in ourselves and steam pressure in a boiler; without a minimal steam pressure the engine will not run and without a minimum of tension we would not be able to perform our daily tasks acceptably. On the other hand, excess of steam pressure results in inefficiency, fuel waste and in extreme cases loss of steam through popping of the safety valve. Likewise when tension is excessive we develop inefficiency, waste of effort through anxiety symptoms and in extreme cases panic occurs, which is likened to the safety valve popping off. Here it is reiterated that mental illness is averted because there is a safety valve, just as a boiler explosion is averted. This is, of course, rather far-fetched, but the fact that it makes tension states intelligible, after a fashion, to even the simplest persons seems to justify its use. This is in accord with accepted principles of psychotherapy which appears in the main to be aimed at promoting understanding and acceptance of the nature of one's illness. In any event, we have found that it is quite useful to have the patient feel that there is a definite plan of treatment and, furthermore, one that he can understand. At the risk of working our poor little analogy to death, we explain the importance of psychotherapy in terms of the steam boiler by saying that it is not enough to relieve steam pressure by blowing off steam periodically, but that in order to promote lasting efficiency the fires must be drawn a bit. Therefore, we must get at the sources of excess tension by examining into the causes, clarifying the origins of basic insecurity and, where possible, showing the relationship between these hidden factors and the present anxiety state. It is important that this "drawing of the fire" by psychotherapy be emphasized because the patient is very likely to believe, otherwise, that the intravenous injection constitutes the whole of therapy, whereas it is only an adjunct to therapy.

It is of interest to note that immediate reactions to this treatment are variable. The commonest statement is, "If I could feel like this all the time it would be wonderful." Others laugh or cry and sometimes smell bad odors, hear crickets or bells, see pleasant pictures and occasionally there is brief diplopia or distortion of vision in which the walls seem to move, etc. The individual reaction tends to be repeated with each injection. Happily, the patient whose symptoms are predominantly gastro-intestinal, usually eats a steak dinner after each treatment.

After the initial treatment the patient is asked to return in five days. He is warned that the immediate feeling of relief may last from two to four days but that the tension symptoms will return before the end of the interval although they will be less intense than before the first treatment. He is quite comfortable at this time and does not care too much if they do return. He is also told that he may not have as good a result as we expect; that the duration and extent of relief obtained is a measure of the severity of his tension and that further treatment will be based upon the result he obtains.

On his return in five days the patient is invariably happy and hopeful. He feels that at long last something is being done for him and in most instances says that he felt better after the treatment than he has felt in years. The degree and duration of relief vary considerably as might be expected. In the mild to moderately severe anxiety states the patient usually reports that he was almost entirely free of symptoms for two days, that on the third and fourth days he felt fairly comfortable but noted a partial return of symptoms and that on the fifth day the symptoms were almost as severe as before treatment. In the more severe cases relief may have lasted only overnight or even only a few hours, but always the patient is anxious to continue treatment and nearly always his attitude toward his illness is improved in that he has been convinced, in some degree at least, that his symptoms are functional rather than organic in nature. Atten-

tion is called to the fact that we are deliberately "overshooting" the duration of the period of greatest relief. This is done in order to insure that the patient repeatedly comes to grips with his symptoms. In this way he gains familiarity and better understanding of the fact that his discomforts are only simple expressions of excess tension and not the dread disease nor harbingers of insanity which he once believed them to be. Thus, at the outset the patient is enabled to adopt a relatively objective attitude toward his own discomfort which is a very favorable factor in subsequent psychotherapy. We have spoken chiefly of somatic expressions of anxiety thus far, but we do not mean to say that only these symptoms are relieved. On the contrary all other anxiety symptoms are relieved quite as effectively as are these of somatic nature.

The patient is now ready for his second treatment which is again given at the end of the psychotherapeutic session. Usually the amount of Sodium Amytal is increased to 2.8 gr., especially in cases where tolerance for barbiturates has been acquired through prolonged use of sedative medication. This must be determined in each instance by the patients immediate reaction to the injection. In most cases it will be found that 2.8 to 3.0 gr. will suffice although some patients may require up to 4.0 gr. to produce the desired relaxation.

Where the initial response was good the interval between the second and third treatment is seven days. In the more severe cases shorter intervals are desirable, but in all cases should be lengthened as rapidly as possible. It will be found that the patient is much more amenable to psychotherapy than one ordinarily expects and that consequently the therapist can proceed at a much more rapid rate than is ordinarily possible. The patient usually reports at this time that his friends, family and business associates have noted a distinct change for the better in his demeanor and says that he can now "take things in stride" which would have upset him badly prior to treatment. He is becoming more self-confident and assertive, though he still has bad moments of relatively minor stress and is not yet fully in control of his tension.

The fourth treatment, given after a 12 day interval, usually finds the patient further improved. Though he still has trouble at times, traceable to some minor stress. He has found by this time that major stresses are still capable of upsetting him badly, but that he soon regains his composure without having to resort to sedation. It is pointed out that the major goal of treatment, the restoration of his own automatic control of tension is now becoming manifest. To those who can understand it, Cannons Theory concerning homeostasis is explained and applied to the case at hand. By this time also, we are usually able to interpret to the patient some of the factors in the development of his anxiety state, which he often finds amazingly simple once they have been clarified.

On his return for the fifth treatment, the patient ordinarily reports that he is getting along quite well and has very few and very mild upsets which do not bother him much. He is surprised to find that he has gone two weeks without help of any kind and begins to feel quite confident of his future ability to handle things for himself. With this favorable development, the interval is increased to three weeks and the patient is told that he is almost ready to "go it on his own," that if he does well at three and four week intervals, then his treatment is completed.

At the termination of treatment, the patient appears to have no difficulty in breaking off whatever transference he may have developed. He is happy to be "back in the saddle again" and has no further use for his therapist. However, he usually knows one or more persons who, he believes, would be benefited by the same treatment and he often succeeds in referring this friend or relation to our office. It is emphasized that physician referral is much preferred, but many come directly on the advice of former patients.

While the above described response to treatment cannot be expected in every instance, it is observed regularly in most milder anxiety states. In the more severe conditions such as the obsessive compulsive states, we cannot expect such a response, of course, but we believe that even in these extremely difficult cases therapy is easier and

more rapid when Sodium Amytal is used in the manner described.

We have further noted that treatment in conditions other than anxiety states is rendered much simpler by the use of Sodium Amytal. For example, we have found that we are able to materially assist the chronic schizophrenic in maintaining an adequate level of social remission. We believe this is possible primarily because the patient feels that something of a positive therapeutic nature is being done for him. Thus, rapport with these very inaccessible patients is greatly facilitated. In a number of instances we have been able to maintain these patients in fairly adequate jobs when failure of adjustment might have been expected. The chronic alcoholic will sometimes, though not often, come regularly for psychotherapy and Sodium Amytal in lieu of drinking. A few occasional alcoholics come for intravenous Sodium Amytal when they feel they are impelled to go on a binge or when they are recovering afterwards. One man comes regularly at monthly intervals for Sodium Amytal. He says it prevents his drinking and costs less.

The headaches of migraine and the post concussion syndrome often respond well, as noted by Susselman, et al. The traumatic neurosis with complicating compensation factors often can be helped considerably, though we have noted that treatment after financial settlement is much more effective than before settlement. Epileptics whose convulsions seem to be related to or adversely affected by tension states are benefited by this therapy in that their response to anti-convulsant drugs seems better when anxiety is brought under control. Also the chronic anxiety states of severe degree, some of which had failed to respond to electroshock therapy, were able to be brought under a fair measure of control, for which the patients have been uniformly grateful. Impotence, where anxiety is a major etiological factor, responds to this treatment and frigidity is often greatly ameliorated. Neurodermatoses may be relieved, even in long-standing cases. The senile neurotic, whose treatment in my hands has otherwise been of little benefit, can be helped more effectively when Sodium Amytal is used. In some cases the juvenile delinquent responds quite well and we have attempted to differentiate between ordinary behavior problems and young psychopaths by this means.

Further, we are able to help patients who suffer from anxiety states and who are ordinarily not amenable to psychotherapy, such as the mental defectives and those with severe language handicap, by using Sodium Amytal as described above. Since our community is, to a large extent, bilingual and we are not, this is of considerable importance.

We cannot emphasize too strongly that the recommended inclusion of intravenous Sodium Amytal in the routine therapy of the anxiety states does not mean that standard diagnostic and psychotherapeutic techniques may be neglected nor abandoned. This point is stressed because we have noted in ourselves a strong tendency on occasion to rely on this simple medium to the neglect of more arduous but necessary psychotherapy.

We have long been aware that our method of therapy carries a very strong element of suggestion and we have often wondered if this variation from standard techniques (i.e. the inclusion of intravenous medication in routine treatment) is truly justified. But always, we are reminded that the results appear to be much better, that the duration of treatment is materially shortened, that the cost of treatment is sharply reduced and, finally, that the therapist is enabled to carry a greater case-load than would otherwise be feasible. This latter consideration is of considerable importance since I am the only practicing psychiatrist in an area larger than many states.

Conclusion

1. A variation from standard psychotherapeutic practices is described and offered as an effective addition to the therapeutic armamentarium.

2. It will be found effective in treating mild anxiety states and useful in other conditions.

3. It should be utilized only as adjuvant to psychotherapy.

Author's Note: Sodium Amytal used in this study is manufactured by Eli Lilly Co., Indianopolis, Ind.

Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE NO. J.H.-8421

Presentation

A 62-year-old white man was admitted to the hospital on February 1, 1950, because of severe pain in the lower back and in both legs. He had had pneumonia and empyema in 1931 for which he was treated with bilateral thoracotomy. In 1935 he sustained a fracture of the left humerus which never united. For it he wore a brace. He may have also injured his low back at that time. In September 1948 he suffered a coronary thrombosis and was hospitalized for 13 days. He received heparin for 5 days and dicumarol for 12 days. It was impossible to keep the prothrombin level depressed, it kept reverting to normal. Recovery from the myocardial infarction was uneventful and he remained well in the interval until January 24, 1950.

On this date he experienced sudden onset of pain in the left leg aggravated by walking. He was seen by a physician who put him to bed and began dicumarol therapy. The anti-coagulant was continued until January 29, but on this occasion difficulty was experienced with a prothrombin level which would drop precipitously to very low values (16 per cent). The pain remitted. On January 30 he was gotten up in a chair and on the following day he began to have pain in the lower back. The pain became progressively worse during the night and radiated down both lower extremities. He was brought to the hospital with the tentative diagnosis of sciatic disorder and to be put in traction.

On admission to the hospital on February 1 he could stand only by markedly flexing the spine, and the pain was least severe while he was in the sitting position. The pain radiated from the lower back down the posterior aspect of both thighs and legs to the ankles. It was greatest in the popliteal regions and was more severe on the left. It was aggravated by all attempted movements of the lower extremities and by coughing or sneezing. There was no muscle spasm, weakness, numbness, nor tingling. His blood pressure was 178/100. Straight leg raising was limited to 10 to 15 degrees bilaterally. There was tenderness along the left saphenous nerve, the patellar and Achilles reflexes were equal and active. The plantar responses were flexor. The peripheral blood contained 19,100 leukocytes per cmm. of which 80 per cent were neutrophiles, 13 per cent lymphocytes, 6 per cent monocytes, and 1 per cent eosinophiles. The prothrombin content was 9 per cent of the control.

On February 2 complete urinary retention developed and numbness and weakness appeared in the toes on the left. On the following day the right foot also felt numb and there was loss of appreciation of pain over the lateral aspects of both feet and of the left leg. The patellar reflexes were still active, but the Achilles reflexes were now diminished, more so on the left. Later that day there was weakness of movement of both feet and paravertebral muscle spasm was found in the lower lumbar region. The hypesthesia was now present in both legs, in the posterior aspects of both thighs, and in the peri-anal region.

A lumbar puncture was done on February 3 with the patient poorly relaxed. The initial pressure was 400 mm. of cerebrospinal fluid and there was no rise in pressure on compression of the jugular veins. The fluid was grossly bloody and contained 264,000 erythrocytes per cmm. Myelography revealed an almost complete subarachnoid block at the fourth lumbar interspace, the pantopaque having been introduced from below this level. The prothrombin content of the peripheral blood on February 3 was 20 per cent. On February 4 there was no change in neurological signs, the prothrombin content was 38 per cent. Even with the utilization of Vitamin K and blood transfusions the prothrombin content could not be raised more than 58 per cent, despite which an exploratory laminectomy (L3-L5) was performed on February 5.

Differential Diagnosis

Dr. John W. Devanney: Referring to the past history I do not believe the pulmonary disease of 20 years ago was in any way related to the present disease nor the fracture of the humerus 15 years before. If he did sustain a back injury at that time, I think the long period of freedom from symptoms rules out any connection to his present difficulty. I think his coronary thrombosis at age of 58 years is a general indication that vasculature was poor.

The onset of the present illness is rather confusing. Evidently his family physician thought he had thrombophlebitis or phlebothrombosis and put him on dicumarol. However, in retrospect we feel that the leg pain heralded the approaching neurologic lesion since the protocol lists no trophic or infectious change in the left leg on admission or subsequently. At the time of admission the referring physician's diagnosis had changed to sciatic disorder.

This confuses our thinking about etiology. If we blame dicumarol for the vascular lesion we face the probability that the lesion preexisted the administration of that drug. Then all we can say is that perhaps the dicumarol aggravated a pre-existing vascular lesion. At the time of admission, February 1, he had bilateral pain localized in what would seem to be the first sacral dermatome bilaterally. The straight leg raising test was also positive.

On February 2 things were moving rapidly. Not only did pain remain in the first sacral dermatomes but paresthesias developed. The ankle jerks were reduced markedly, if they were not absent. Urinary retention had appeared which pointed to involvement of the third and fourth sacral levels and sensory changes were present in all segments caudal to the first sacral distribution.

At this stage the case presented aspects of a rapidly progressing intraspinal lesion. This recognition, I feel, is the most important factor of this discussion. Cases of this sort are true neurosurgical emergencies and must be recognized early and therapy begun immediately if good results are to be attained.

We know that brain, cord, or peripheral nervous tissue even if gradually compressed over a long period of time can take much punishment before evidences of malfunction appear. When slowly progressive lesions are decompressed the nervous tissue often resumes normal function though it has been under stress for a long time. However, this is certainly not true for acute compressions of nervous tissue whether they be traumatic or from any other cause. I repeat, these lesions must be recognized early and definitive therapy instituted as soon as possible, because in acute lesions there is evidence of a relationship between the time that pressure exists and the amount of damage that is done. The possibility for return of function is inversely related to the duration of compression.

By February 2 our anatomical diagnosis was well developed, but first I would like to localize the lesion more accurately. We must remember the difference between vertebral and neural levels in the spine. I say neural rather than cord levels because in the lower spine we are below the termination of the cord and in the area of the cauda equina. This is where I believe the lesion was located, at a point where the first sacral nerves were compressed early, then as the lesion progressed, all spinal nerves caudal to this were involved. This points to the lower lumbar region, say at the level of the fourth or fifth lumbar vertebra.

At times it is difficult to distinguish, on the basis of the neurologic examination, between a caudal lesion of this sort and a lesion of the conus medularis; the first sacral cord segment as you know is located at about the D-12 vertebral level. I do not think the latter likely because such a rapidly progressing lesion as this one was, if it were located at the twelfth dorsal vertebra, would have also involved the lumbar spinal nerves as well as those already mentioned.

In any event we have diagnostic aids that are immediately indicated when a space-consuming spinal lesion is suspected, lumbar puncture and myelography. Here the lumbar puncture revealed a fairly complete manometric block and a high initial pressure. There were counted 264,000 red blood cells per cmm. Myelography confirmed our localization at the fourth lumbar interspace, the pantopaque having been introduced below this level.

Confronted with a diagnosis of a space-consuming intraspinal lesion, accurately localized, and with both the story and myelogram pointing to extramedullary or extradural lesion, decompression was indicated as soon as possible. The physicians in this case were confronted with a difficult choice. On the basis of what we have said immediate operation was indicated. However, this patient was evidently not in good condition, and because of his dicumarol or for some other reason his prothrombin concentration was markedly depressed and remained so.

I imagine that the neurosurgeon was champing at the bit while awaiting the attempts to correct the blood deficiency. I believe his decision to go ahead despite this, after a two-day delay, was justified. If he did not operate he was likely to have a paraplegic patient, and paraplegics in this pa-

tient's age group do not live very long. If I were faced with this situation I would do exactly as was done: operate with plenty of blood on hand and be as meticulous as possible with hemostasis during the procedure. We know that we are dealing with a space-consuming extradural lesion whose lowest extent is at the L-4—L-5 interspace. Whatever its nature surgical exploration and decompression was indicated.

For completeness of diagnosis we must consider the nature of the lesion. Despite the fact that often in atypical lesions of the central nervous system one may be mistaken in this regard, it is consoling to believe that you know the type of lesion you are attacking. Let us consider differential diagnosis. The most frequent space-consuming lesion in this area is ruptured intervertebral disk, and though this story is rather atypical for disk, a large bar disk could cause all symptoms and signs noted. However, I believe the timing and method of onset rule out this possibility.

Tumor, the second most likely cause from a statistical point of view, is unlikely. Tumors do not grow this fast, although bleeding into a tumor sometimes calls it to our attention more quickly than otherwise. Trauma is mentioned only for completeness; there is nothing in the history or roentgen reports to suggest it.

Let us consider extradural abscess. Here the timing fits well; it is usually a rapidly fulminating affair. But where then was our primary source? Two-thirds of these are considered to originate on an embolic basis, one-third from direct extension from a lesion close by, as for example, following trauma, or from Pott's disease and that sort of thing. We really have no evidence that this man had infection anywhere that might have involved the extradural space either by direct extension or via the blood. On the other hand, the white blood count was elevated, and the differential count shifted to the left. Extradural abscess must be considered a possibility.

A word of caution concerning spinal puncture if abscess is a consideration. You can readily see the danger of going through an abscess cavity and carrying pus via the needle into the subarachnoid space and also that leaving a puncture hole in the dura may encourage spread of pus in the other direction. This danger is present even though the abscess be higher than the level of the tap, for there is likelihood that due to gravity and lack of compartmentation of the extradural space, pus may migrate from the abscess into the lumbar area. In such cases it is a wise precaution to try to strike the lamina with the needle and to withdraw the stilette and watch for pus to appear. You may have to wait a bit as pus is often quite viscid and under low pressure. If no pus is found, proceed with the top, at say 2 mm. advancements, removing the stilette and checking repeatedly until the dura is punctured or pus appears. In this latter case, stop immediately as you have your diagnosis.

The rarest lesion in this area is the one on which I am going to rest my case, that is a non-traumatic extradural hematoma. Its course is very rapid, that fits our case as does the blood in the cerebrospinal fluid, for there is usually some subarachnoid bleeding too. The article by Kaplan and Denker[1] which contains a thorough survey of the literature records only 8 cases reported up to 1949; they added 2 cases of their own, observed at Bellevue Hospital within 6 months. It is pertinent to remark here that statistics don't make any difference to the person who has the lesion, for though the trouble may rarely occur for the patient, it is as though the incidence were 100 per cent.

A few important points from the paper by Kaplan and Denker, particularly to stress the catastrophic effect of acute compression of the spinal cord, even for short periods of time, which emphasizes the importance of early and accurate diagnosis. One of their cases was not diagnosed or even hospitalized for 7 days after the onset of symptoms. Operation was performed on the eighth day. No recovery from complete paraplegia was noted in 15 months of available follow-up. In another case 5 days intervened between onset of symptoms and admission to the hospital. Operation was performed on the sixth day, and there was no recovery in 8 months of follow-up.

One of the two patients treated at Bellevue had the lesion between D-5 and D-6,

the other at D-12. Of the other 8 garnered from the literature, not all were treated surgically, but of those who were, only 3 recovered. While it was not stated specifically, I am sure these 3 patients were operated on early.

They also mentioned that in 8 of the 10 cases onset accompanied episodes of increased intraspinal pressure as may occur in straining at stool, vomiting, etc. In 5 of the 10 cases paraplegia was complete in 3 hours. This is certainly very rapid, faster than in our case, but he too moved along rapidly.

To sum up, I believe that this man had a non-traumatic extradural hematoma. My second choice would be epidural abscess, and thirdly, hemorrhage into a tumor. Fourth, and I believe most unlikely, a ruptured intervertebral disk. I believe the most important factor to be learned from this case is to make the diagnosis of acute, space-consuming intraspinal lesion promptly, recognize it as an emergency, and act accordingly.

Dr. Daniel Osher: The roentgen studies revealed there was evidence of an old and slight compression fracture at L-4. On myelography we couldn't get more than a little trickle of pantopaque past L-4. There appeared to be a mass compressing from posteriorly on these films, resulting in practically a complete block.

Discussion of Pathology

Dr. Osher: Dr. Devanney's discussion has caused a return of the anxiety I had at the time I saw this man on February 3 when he gave the history of pain in his calf ten days before, when he was started on dicumarol. The man had some swelling of the calf and some tenderness. It was not an acute febrile type of thrombophlebitis, but apparently some sort of phlebothrombosis and with dicumarol and bed rest relief of symptoms occurred within 24 hours.

He was continued on dicumarol and then about a week later he had the sudden onset of severe low back and bilateral flank pain which seemed to represent a new entity. I do not believe that the previous pain was related to this condition. The next day he had bilateral leg pain and then that same evening developed weakness and numbness of both feet and difficulty in voiding. Pain was the outstanding symptom; this man was in agony. He couldn't get in a comfortable position and if he made any movement of the extremity he would cry out. Analgesics and narcotics had very little effect on him.

On February 3 when I first saw him he had active knee jerks, but these were soon entirely absent and he had lost sacral sensation bilaterally from S-1 through S-5. So I think we had rather definite localization of the lesion to the lower lumbar region.

On spinal tap there was an initial pressure of 400 mm., but he was tense and poorly relaxed. He had complete block on manometric testing though it was difficult to evaluate adequately because the fluid was grossly bloody and finally clotted in the needle. Immediately 2 cc. of pantopaque was injected, and fluoroscopy and X-ray showed an almost complete block at L4-5 interspace with some of the oil going by on the left. These plates also showed the questionable old compression fracture of L-4.

I saw him from 12 to 2 P.M. on February 3 and suggested immediate surgery for the reasons that Dr. Devanney gave; then they brought me up short with this very low prothrombin concentration. It was decided to try to build up the prothrombin time as quickly as possible with blood transfusions and vitamin K and to defer surgery for a day or so. Unfortunately the patient responded slowly and inadequately. On February 4 my note read, "Awaiting increase of prothrombin time. He continues to have marked spasms in his legs—these shooting pains—and he has very little movement of his toes and ankles bilaterally. The sensory level is unchanged." He had an in dwelling urethral catheter. The next morning (February 5) with the prothrombin concentration up to 38 per cent it was decided to operate.

Dr. Charles D. Aring: The students in their written discussions did not agree with Dr. Devanney's diagnosis. Sixteen thought that the trouble was a cauda equina tumor, some of them postulating that it extended above the cauda equina into the spinal cord. They were divided as to whether it was a

primary or a metastatic neoplasm. Another 13 believed that it was a massive herniation of an intervertebral disc. Two thought that it was hematomyelia, one man suggesting that it arose from an angiomatous lesion. Two students made a diagnosis of epidural abscess. Therefore, the student group unanimously disagrees with the discussor as to the nature of the lesion, an unique experience for these conferences. I would like to hear some discussion, particularly from the students.

Student: It was my impression from reading, that in hemorrhagic lesions of the epidural space the blood spreads upward or downward and therefore a clinical picture of a more diffuse pattern or broader range would be expected than we see in our patient.

Doctor Osher: The epidural space in the skull is very limited; there is adherence of the dura to the bone at the suture lines and at times the dura is densely adherent to the bone elsewhere, especially in older people. In the spinal canal the situation is different. The epidural space is not tightly adherent; in fact, it's so loose that it is possible to do an air epidurogram, that is inject air into the epidural space under pressure and take pictures of this air spreading through the epidural space all the way up to the foramen magnum. So you can see how loosely applied this dura is. This supports the suggestion that epidural bleeding there would spread. The question is how far is it going to spread and what limits the spread? Clinically, we know that these lesions do not spread all the way up or down. From studies of epidural abscesses we know that spread may be wide, in fact an epidural abscess of the entire spinal vertebral column has been reported. However, it is usual for epidural abscess to be localized over a distance of four or five laminae, so there is limitation to this spreading factor. I don't know why. Our patient's signs did indicate a certain amount of spreading. From a more focal lesion at onset there was indicated spread to the entire lower cauda equina in the lumbar sac.

Student: Was the cerebrospinal fluid spun down to see whether it was xanthrochromic?

Doctor Osher: We didn't get much fluid because of the rapid clotting, however, it contained 5000 mg. per cent protein, and the supernatant fluid was dark yellow.

Dr. J. Parke Biehl: How do you feel about putting oil into the subarachnoid space when there is a possibility that there is a traumatic tap? Might not the oil get into a vein?

Doctor Osher: I think that possibility exists; I don't believe that I thought of it at the time. The fluid that I obtained on lumbar puncture was suggestive enough of bloody cerebrospinal fluid that I wasn't worried about being in a vein. I would not inject pantopaque if I thought I was dealing with a purely bloody tap.

Student: In Walshe's[2] discussion of spinal lesions he notes that hemorrhage plays no significant role.

Doctor Osher: Well, I agree with Walshe. In fact, primary hemorrhage was one of my later diagnostic choices in our case. I knew that there was hemorrhage present; the problem was what was the hemorrhage due to? I thought it was related to the low prothrombin. Then, why did he choose this particular spot to bleed in and nowhere else? This brings up the diagnostic possibilities of other lesions that might have caused hemorrhage secondarily.

Student: If this man did have a hemorrhage, could this hemorrhage be responsible for all of his signs and symptoms?

Doctor Osher: We do know that extradural bleeding can cause compression of the spinal cord.

Student: Wasn't the presence of blood in the cerebrospinal fluid more in favor of intraspinal lesion or lesion in the subarachnoid space rather than extradural hematoma?

Doctor Aring: Couldn't it be carried in with the needle?

Dr. Osher: It is possible to carry blood in with the needle, transmitting it from extra or subdural space to subarachnoid space, or on the other hand tapping cerebrospinal fluid into on extradural collection of blood. My preoperative diagnosis was an expanding lesion within the spinal canal at L-4, tumor, ruptured disc, or extradural hemorrhage.

The spinous process and lamina of L-4 was removed. This revealed a purplish, bulging mass in the epidural region. At first this was thought to be distended dura. However, after the removal of the ligamentum flavum on the right between L-4 and 5 it was seen that there was a large, purplish, black epidural clot. This was gradually removed by means of suction and the use of pituitary rongeurs, and it was seen that the dural sac had been depressed and pushed to the left at this site for about 1 inch. As the clot was removed, the dural sac expanded and filled the space. The clot was primarily posterior and on the left side. A small bit of the upper border of the right side of the lamina of L-5 was then removed and the dura retracted medially. The lower portion of the clot was removed at this point. This extended about half way beneath the lamina of L-5.

Attention was then directed to the upper portion of the clot, and a hemilaminectomy was done on the right side of L-3, and in addition, the ligamentum flavum between L-2 and 3 was removed. Again retraction of dura facilitated the sucking out of the clot. The upper extent of the clot was not visualized. The dura had in the meantime expanded to fill the space and pulsations could be palpated. I did not feel it wise to explore further, so a small rubber catheter was passed upward for a distance of about 5 cm. without any resistance. A small amount of saline was irrigated through this catheter. Then I inserted a number 18 spinal needle into the dural sac underlying the exposure at L-4 and attached a manometer. The pressure measured 200 mm. of water and the fluid was pink. Jugular compression caused a prompt and adequate rise in pressure, and it was believed that there was no further obstruction.

Then a tiny nubbin of arachnoid herniated out through the needle puncture in the dura. This is important because it correlates with what might happen after spinal puncture. A large amount of blood-tinged cerebrospinal fluid gushed forth which gradually cleared. A piece of jell foam substance was placed over this hole to stop the cerebrospinal fluid leak. I've seen this sequence of events many times before—the leaking fluid coming from the needle puncture wound. Sometimes the little nubbin of arachnoid which sticks out through the hole keeps oozing fluid. Perhaps this may be one of the reasons for spinal puncture sequellae, headache on assuming uprightness and pain in the lower extremities.

The wound was thoroughly irrigated and explored for evidence of bleeding. The dura was retracted medially toward the left. The fourth lumbar root was seen to be lying well anterior in the vertebral canal. There was no evidence of tumor or displaced disc. We put in a lot of jell foam and watched carefully for further bleeding. It did not occur. The patient got 1000 cc. of fluid during the procedure and he left the operating room in good condition. Study of the tissues removed at operation revealed nothing but organized hematoma.

Clinical Diagnosis

Expanding lesion of spinal canal at the fourth lumbar vertebra; either neoplasm, ruptured disc, or extradural hemorrhage.

Coronary heart disease with old myocardial infarction.

Dr. Devanney's Diagnosis

Extradural hemorrhage, non-traumatic, at the fourth lumbar vertebra.

Anatomical Diagnosis

Extradural hemorrhage beneath the third, fourth, and fifth lumbar vertebrae, probably abetted by low prothrombin concentration in the blood induced with dicumarol.

Doctor Osher: This case reiterates the emergency nature of spinal block. We were loath to let this man go as long as we did, and I think the delay was reflected in his slow progress. He had a lot of trouble postoperatively. After the operation of February 5 his pains improved. On February 7 there was improved movement of right foot and toes and the left foot was slightly improved. The sensory level remained about the same.

We watched carefully for recurrence of bleeding; Prothrombin concentration rose slowly; it was up to about 50 per cent two days after operation and shortly it became 70. He was much improved on the third post-operative day. He had some painful spasms but they had almost disappeared. Motor and sensory function was improving

in the feet. He continued to improve and was discharged on March 3, approximately a month after operation. He was able to walk without support, but the left foot was still weak. His knee jerks were active. His right ankle jerk was weak; the left was absent. He had a flexor plantar response on the right and no response on the left. The latter was a peripheral phenomena rather than indicating pyramidal damage. The abdominal skin reflexes were present. He had decreased sensation in the first sacral segment on the left; there was complete return in the left lower sacral segments. On the right there was a small area of hypalgesia in a portion of the first sacral, and sensation was diminished in the other sacral segments on the right. He still could not void without a catheter. The remainder of the neurological examination was normal except for weakness of the toe movements on the left.

We discharged the patient on catheter drainage. He was attended by a urologist at home and each time the catheter was removed he could not void. The day before his discharge from the hospital a cystometrogram was done which showed evidence of an atonic neurogenic bladder, which is the type of bladder one would expect with a cauda equina lesion. At home he continued to improve, but he required the catheter. One month later he returned to the hospital and a cystometrogram was done which showed a fairly normal pressure curve with involuntary emptying contractions associated with voiding around the catheter and a marked rise in intravesicle pressure after 300 cc. of water was put in. This represented a considerable change from the previous atonic bladder. We couldn't understand that with this man's improvement in neurological status, why he couldn't void. The second cystometrogram showed that he had something other than a neurogenic atonic bladder. In other words, he had good muscle contraction and he had sensation. He was readmitted to the hospital and a transurethral prostatic resection was done, and he has been able to void normally ever since. This experience stresses the importance of cystometry which is a neglected part of our diagnostic armamentarium. At the present time, a little over a year after operation, this man is well. He has the sensation like a rubber ball being present in his rectum. He has occasional causalgic-like pains in the toes of his left foot. I haven't examined him for many months, but his neurologic status is practically normal now. His Achilles reflexes have reappeared.

Doctor Aring: Did he have prostatic hypertrophy, or did the operation just weaken the median bar?

Doctor Osher: He had a bladder stone. It is well known that patients with neurogenic bladder tend to get calculi in the bladder. There was only moderate enlargement of the prostate (lateral lobes and the median commissure) but transurethral resection of the prostate and removal of the stone resulted in normal urinary function.

Doctor Aring: Well, this illness was a fascinating problem. I would like to accent what both Drs. Devanney and Osher said about the need to attack quickly cord or cauda lesions that come on suddenly and advance rapidly. If cord or cauda compression develops slowly, then the tissues have a better chance. With a quickly occurring compression the nervous tissue is not afforded the opportunity to reach an equilibrium and we have to move fast. I am sure Dr. Osher was most impatient while his colleagues were trying to raise the prothrombin concentration. He wanted to relieve this compression because he knew that these acute compressions may result in long-standing or permanent residuals.

*Dr. Helen Glueck:** I heard about this patient in a "cloak-room consultation." I think the problem is why he developed a spinal epidural hemorrhage which is certainly a very rare complication with anticoagulant therapy. I have been unable to find a similar case. Six cases of hemorrhage in the cord have been reported by Nichols in patients who had thrombophlebitis and received anticoagulants and on whom a lumbar block with procaine was done in the attempt to relieve pain. In these patients transverse myelitis due to hemorrhage occurred following the lumbar block, possibly due to oozing external to the dura from the needle point and thence compression with embarrassment of blood supply to and from the cord. Nichols and others feel so strongly about this that certain physicians using anticoagulants advise either a block or anticoagulant therapy but not both. Certainly in all the patients whom we have treated with myocardial infarction with dicumarol in this hospital, well over 200 by now, we have not seen spontaneous hemorrhage in or about the cord as occurred in this patient.

Another point that was extremely interesting and troublesome in this patient was the failure to respond to the usual regime employed to raise the prothrombin concentration to more normal levels quickly. I don't remember how many transfusions

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the patient had, but there were quite a few—I think three, in an attempt to raise the prothrombin level —and he was given a lot of vitamin K without response. Recent clinical investigation has shown that Vitamin K-1 oxide which is a difficult substance to use because it oxidizes quickly and must be emulsified, will raise the prothrombin concentration much more rapidly than synthetic vitamin K which was used in this patient.

A point I would like to bring up in this discussion is the effect of anticoagulant therapy on the central nervous system. This is a very important problem which has been neglected by people working in this field. It has been shown distinctly, for instance, that in the pre-penicillin days when patients having subacute bacterial endocarditis of the streptococcal viridans group were given dicumarol for the cerebral embolic phenomena which occurred; the incidence of cerebral hemorrhage was high. It is well known that hemorrhage can occur with subacute bacterial endocarditis anyhow, but it raises again the whole question, "What are the effects of dicumarol on brain tissue?"

Certain of the newer anticoagulants, particularly tromexin may have an advantage since its effect is short lived, not more than 24 hours. Within 12 to 24 hours after administration of tromexin the prothrombin time is back to normal.

Finally, I want to say a word about hemorrhage with dicumarol. Although we have the so-called "hemorrhagic level" and the so-called "safe level" and are not supposed to see hemorrhage unless the prothrombin concentration is less than 10 per cent of normal, I have seen hemorrhage with dicumarol in which the prothrombin concentration was 40 per cent of normal, and no hemorrhage when the concentration was so low it could not be measured. Statistically you may be safe, but occasionally there is the patient in whom other factors, possibly capillary, complicate the whole picture and in whom hemorrhage does occur with levels of 30 and 40 per cent of normal concentration.

Doctor Devanney: I might say I've seen a subdural hemorrhage removed in a hemophiliac who had received repeated transfusions for three days prior to operation, and there was less bleeding than in the average extradural exploration.

Doctor Glueck: About surgery in the face of dicumarol prothrombin deficiency, I have seen an open reduction of the arm where it was absolutely urgent for the orthopedist to operate in which the prothrombin concentration was about 50 per cent of normal. I think it's safe to operate when the prothrombin levels are 50 or 60 per cent of normal. If you transfuse the patient during surgery, usually you can "get away with it" at those levels. It's not to be encouraged of course. In emergencies it may have to be done.

Dr. Lennart Tegner: What about chain transfusions with whole blood as is done in the leukemias?

Dr. Glueck: Done in children for instance with the RH factor? I can't answer that. Most people say that 1000 cc. of blood will raise the prothrombin concentration rapidly enough. Of course, what's so interesting is that the next day it will fall again.

Doctor Biehl: Must the transfused blood be fresh or may it have been stored?

Doctor Glueck: There is some argument about this, which raises another point. Quick has shown that there are probably two factors in prothrombin, one which is destroyed by dicumarol and one which is destroyed by standing, but if you mix the two together you get a normal prothrombin time, so you do not necessarily have to use fresh blood.

Dr. Murray Sheldon: I wonder if you would comment on parodoxical antiprothrombin effect of large doses of commercial vitamin K?

Doctor Glueck: I can't say much about it except that Edgar Allen who has had great experience with dicumarol and vitamin K has pointed out that the synthetic vitamin K which we use, when given in doses over 60 mg. a day for several days, may actually depress the liver somewhat so that it cannot produce prothrombin.

It is to be hoped that certain difficulties in the non-soluble vitamin K preparations can be remedied. At present these preparations are sensitive to light and since they are insoluble in water must be emulsified in alcohol. Nevertheless they are much more active than the aqueous vitamin K preparations and should prove useful in treating dicumarol induced vitamin K deficiency.

Doctor Aring: I would like to call attention to an anatomical point about the circulation of the spinal cord that makes sudden extradural compression serious. The upper part of the cord receives its blood supply via the anterior and posterior spinal arteries, branches of the vertebral arteries. These vessels become attenuated as they approach the mid-thoracic region, and caudally the lateral spinal arteries coming from the intercostal, lumbar, and lateral sacral arteries are responsible for the maintenance of blood supply to the spinal cord. These vessels enter the cord via the roots of spinal nerves through the intervertebral foramina and, therefore, must traverse the extradural space. If there is encroachment on extradural space, the arterial supply to the cord (to say nothing of the venous return) is embarrassed. This, I should think, is more often the mechanism of transverse myelitis than is hemorrhage into the cord itself. If the rich venous plexus in the extradural space begins to bleed and take up space, compression of some degree is likely to occur. Infection, granuloma, or neoplasm in the extradural space carry the same hazard.

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Psychiatry and Religion

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Recently I met a clergyman who posed an interesting question. We had never met before but since we are both basically interested in the same thing—people and their problems—we had a good deal in common. In the course of our conversation the clergyman asked this startling question. "On many occasions I have told men and women who have come to me with problems that they must seek the aid of a psychiatrist. Yet I have never heard of a psychiatrist who has returned the compliment and sent me a patient for spiritual help. Why is this so?"

Before even attempting an answer, there must be a definition of terms so that references will have an acknowledged meaning. What does *religion* mean to a psychiatrist? Perhaps an anecdote will illustrate my own feelings. When I was still an interne, fresh out of school and overwhelmed by enthusiasm, I had a powerful experience. I was rushing down a hospital corridor to the delivery room. A woman was in labor and I was to deliver the baby. Suddenly a nurse stopped me. Despite my protests, she pulled me into a room to examine a patient I had worked with previously. He was a cardiac and I was able to determine very quickly that he had died. There was nothing I could do but pronounce him dead. Then, almost without stopping, I continued my flight down the hall to the expectant mother. In less than fifteen minutes I had helped to bring a new born babe into the world. Imagine, the end and the start of a life almost simultaneously . . . the alpha and omega of living in a quarter of an hour.

That impression has never faded. I had studied to master the mechanics of life. I could help people in and out of life. But I could never understand its mysteries or meanings. I could never answer the "why."

From this, to me religion has become the continuum of man to man, of the animate to the inanimate, of all things to all things else, and all of it bound together by the mortar of love. From this continuum, this unity grow ethics, morals, value standards, deeper meanings. From love grow humility, awe, reverence and, in turn, their resultant outgrowths of faith, belief, hope, justice, and mercy.

Throughout the ages, time and time again man has reaffirmed his own profound insights in telling phrases. But so much of this has fallen on immature minds. Immature minds cannot love. They cannot give. They cannot see things in relation to the totality of all things. Albert Einstein expresses this in his book "The Universe of Dr. Einstein" with unusual clarity.

> "The most beautiful and the most powerful emotion we can experience is the sensation of the mystical. It is the sower of all true science. He to whom this emotion is a stranger, who can no longer wonder and stand rapt in awe, is as good as dead. To know that what is impenetrable to us really exists, manifesting itself in the highest wisdom and the most radiant beauty which our dull faculties can comprehend only in their most primitive form, this is knowledge. This feeling is at the center of true religiousness.
>
> "My religion consists of an humble admiration of the illimitable superior spirit who reveals himself in the slightest details we are able to perceive with our frail and feeble minds. That deeply emotional conviction of the presence of a superior reasoning power which is revealed in the incomprehensible universe forms my idea of God."

The psychiatrist can subscribe to this pronouncement.

As for psychiatry, it is simply that specialty in medicine which deals with the diseases of the mind and the emotions, with personality maladjustments and disturbances.

Within psychiatry there are still further specializations. Psychoanalysis is one such and the hub of much heated discussion. This is basically a method of treatment and a body of knowledge first introduced by Sigmund Freud whose concepts opened a whole new approach to self-knowledge and self-understanding.

Until Freud, we were both afraid and unable to see ourselves as we truly are. Through psychoanalysis we were, in a sense, first introduced to ourselves. And it was

not a pleasant sight by the then current standards. We saw that as babies we were not sexless cherubs or angels, but rather emotional beings with demands, frustrations, angers, conflicts, guilts, hostilities, jealousies, hates, desires for murder and incest. As a result of Freud's studies, mental symptoms began to have new meanings and much was now explainable in the light of Freud's concept of the unconscious. Mental and emotional disturbances ceased to be strange phenomena explainable in terms of devils or demons. It now became necessary to think of them as emotional arrests in the course of a personality's development.

To a lesser or greater degree, all people suffer such arrests and much of our adult life is governed by these shadows of childishness. We are hampered by them as we strive toward maturity. Psychoanalysis concerns itself with the treatment of these arrested emotional developments. It tries to produce as great a balance as possible between the component forces and aspects of a personality and so prepare for greater harmony with the subtle, universal balances of life as set forth in the great religious goals of mankind.

What conflict can there be between a mature religion which aims at man's maturing and a dynamic psychiatry which also aims at man's maturing? The central aim of life must be the maturation of man!

Through psychiatry we learn how to know, to face, and to accept ourselves. In this knowledge much of the energy that we once diverted to struggles within the component parts of our personality can be directed to the working out of a better harmony between the self and the environment. The energy thus freed can be applied to the process of maturing and self-realization.

At best, the psychiatrist is an artisan at work attempting to free these energies. But it is only through a religious orientation that he can become an *Artist*. Religion alone can provide him with the direction and orientation without which he can never become a true "physician of the soul," to use Plato's phrase.

Psychiatry and religion deal with emotions and feelings. In that, they meet on a common ground and, in a mature way, theirs is a common purpose. From religion we receive such universal truths as: Love thy neighbor as thyself. This is a mature commandment. But you cannot love your neighbor if you hate yourself. Adults who are immature cannot love. This is the prime symptom of immaturity. They actually hate themselves.

How does an immature person hate himself? The same emotional arrests we spoke about before manifest themselves in adulthood in negatives; in intolerance, stubbornness, spitefulness, unreasonableness, malicious gossiping, etc., any of the unnumbered partial suicides we daily commit. Psychiatry attempts to teach proper self-love, self-respect. Such love necessarily overflows to our neighbors and thus we are truly freed to love them as ourselves. As a matter of fact, most of the practice of psychiatry could be described as efforts to assist individuals to better control their aggressive urges, their tendencies to hate and to hurt.

Through such love God is brought down from heaven to earth and the old admonition to "beware of the man whose God is in heaven only" takes on new meaning.

Through psychiatry a man's emotions can be turned in a mature way to God. His religious experience then becomes proportional to his emotional development. An immature man cannot have a mature God concept. We equate God with Love, the highest manifestation of maturity. In loving we have a way of living. Such a religion becomes a constructive way of life for the home, the school, the community, the world.

There is no essential conflict between psychiatry and religion. As a matter of fact, the unity of their purposes makes such conflict almost a contradiction of terms. But what of psychiatrists and clergymen? Some psychiatrists can only see the superstition of religion, its bigotry, narrowness, its mistakes and excesses, its authoritarianism. In this, they are blind to the true meaning of religion.

On the other hand there are many clergymen who see in psychiatry only license for wickedness and ungodliness, a bitter projection of their own immaturities, actually an anti-religious force. In this, they too, wear blinders. A mature clergyman who under-

stands nature and human beings in the light of psychiatric development aims with the psychiatrist at liberating the constructive forces in a personality and at drawing people into fellowship.

With this we can return to the question asked by my clergyman friend. The answer has been stated succinctly by a rabbi. A. J. Heschel has written, "Piety is an attitude towards all of reality. The pious man is alert to the dignity of every human being . . ."

Here the cleric and the psychiatrist meet on common ground.

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